# THE ROLE OF HYPERTENSION

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IT HAS BEEN STATED, on good authority, for many years, that hypertension is a symptom only, an insignificant manifestation of an underlying vascular disease which proceeds inexorably regardless of the level of blood pressure. Therefore, reduction of blood pressure, according to these authorities, is not a rational procedure because the vascular damage proceeds nevertheless. Such a nihilistic attitude was quite permissible 20 years ago when there were no effective methods for reducing and controlling hypertension. The whole question was academic anyway, and if physicians could be dissuaded from giving worthless and presumably dangerous medications, so much the better.

Today, however, the situation is considerably different. We now have antihypertensive agents, or combinations of these, which are able to reduce and maintain the blood pressure often into the normotensive range. For example, in a large-scale cooperative study on antihypertensive drugs in the Veterans Administration, the results, as yet unpublished, indicate that a nontoxic combination of available drugs has reduced the average diastolic blood pressure of a large group of mild and moderate hypertensive patients to below 90 millimeters Hg—that is, into the normal range.

This was accomplished without significant discomfort to the patient and by simple rule of thumb methods of dosage and administration. The study, rigidly controlled by double blind placebo technics, leaves no doubt that methods for controlling the blood pressure at least in the less severe cases, have come of age and are available to any physician who cares to use them. This advance makes the question of the significance of the high blood pressure itself assume a new importance. We must seriously seek an answer now as to whether the presence of a high pressure in the arterial tree is of itself harmful or of no consequence. I have tried to find factual evidence for the opinion that the level of blood pressure is of no consequence. I could find very little.

Usually the opinion was expressed without any factual data to support it, or else it was based on a small personal experience of one physician or one small clinic. In the latter, no attempt was made to differentiate between casual and basal blood pressure; and we now know that a fair number of hypertensive patients may be quite labile, exhibiting much higher blood pressures when the doctor records them in his office or in the clinic than at other times. Failure to differentiate these labile patients from the less labile leads to hopeless confusion when one attempts to correlate blood pressure and longevity, particularly if the series is small.

The life insurance companies some years ago compiled their data on the relationship between the level of blood pressure at the time of the initial insurance examination and subsequent life expectancy. This was a cooperative study involving 20 of the largest insurance companies of North America, and it included hundreds of thousands of individuals.

The results of that study show that in regard to systolic blood pressure there is a smooth curve correlation between

the level of systolic blood pressure and the subsequent life expectancy—which, incidentally, pays no attention to the so-called borderline or normality of 140 millimeters of mercury.

With regard to diastolic pressure there also is such a relationship, which proceeds down to about 88 millimeters of mercury. Thereafter the data suggest that patients below that level have a different condition and have a much better life expectancy.

Since hypertension is a slowly progressing disease in most individuals, it is not yet possible to say with finality that reduction of blood pressure with antihypertensive agents prolongs life. It will be five, ten, or even more years before that evidence is in. The previous speaker said we have plenty of time, but I do not think we have so much time.

There is one form of hypertension where the rate of progression is so rapid that even short-term studies under five years can disclose significant differences.

That condition is malignant hypertension. In an untreated series of 146 patients the survival at the end of one year was 20 per cent and at the end of three years was 9 per cent. In our own series of 64 cases of treated hypertension, there was a 65 per cent survival at the end of one year and about 48 per cent at the end of three years. The experience of other investigators, both in this country and in Great Britain, has been similar; namely, that reduction of blood pressure has prolonged life significantly even in this most severe type of hypertension where vascular damage is already far advanced.

Let us turn now to a consideration of the relationship between hypertension and atherosclerosis. The high incidence of strokes due to cerebral atherosclerosis and thrombosis and hemorrhage is so common in hypertensive patients that it needs no further documentation. It is less well appreciated that coronary atherosclerosis and myocardial infarction are more prevalent in the hypertensive than in the normotensive population. In one study of over one thousand cases of myocardial infarction it was found that the incidence of coronary occlusion was five times more common in hypertensive than in normotensive men, and twenty times more common in hypertensive as contrasted to normotensive women. In the carefully evaluated data from the Framingham study, hypertension was found to correlate significantly with the incidence of coronary artery disease.

Obviously some factors are operative in the hypertensive patient which tend to accelerate and promote the development of atherosclerosis. Hypertension may not cause atherosclerosis but it provides a fertile soil for the development of atherosclerosis. This factor, accelerating atherosclerosis, could be the elevation of blood pressure per se, or some other unknown and mysterious factor which operates entirely independently of the blood pressure.

Evidence on this question can be deduced from some interesting experiments of nature. There are certain types of congenital heart disease such as patent ductus arteriosus with right to left shunt, or large interventricular septal defects where the pulmonary arterial pressure more than quadruples its normal value. Occasionally these patients survive to early adult life; and when they do they are commonly found at autopsy to have extensive atherosclerosis not of the systemic arteries, but of the pulmonary arterial tree.

Now, if the hypertension itself is of no consequence, is it not strange that when hypertension exists in the systemic circulation atherosclerosis is accelerated there and spares the pulmonary vessels? But when hypertension is limited to the pulmonary circulation, atherosclerosis develops in the pulmonary arteries but spares the systemic vessels. It seems that one can fit the facts much better by

postulating that it is the elevated blood pressure per se which accelerates the formation of atherosclerotic plaques.

Another critical experiment of nature is exemplified in the hypertension produced by coarctation of the aorta. Here, because of the constriction in the descending thoracic aorta, hypertension is present in the upper half of the body, but below the constriction the blood pressure is usually normal. Thus, if it is the elevation of blood pressure which accelerates atherosclerosis, one would expect to find more plaque formation above the coarctation in the region of elevated blood pressure than below it in the region of normal blood pressure. And indeed there is evidence to substantiate this expectation.

Time does not permit me to go into the large and growing body of evidence which suggests that high blood pressure is bad for all arterial blood vessels, not only in facilitating the development of atherosclerosis, but also of arteriolosclerosis and necrosis. The evidence also is good that hypertension imposes a heavy load on the heart leading first to hypertrophy and then to dilation and failure.

If this indeed be true, that it is the elevation of blood pressure which is the major cause of the cardiovascular damage in hypertension (and my reading of the available evidence indicates more strongly that it is than that it is not), and since reduction of blood pressure can now be readily achieved in most patients, then the problem of blood pressure control has become an important issue in preventive medicine. No antihypertensive drug can dissolve a fibrous and calcified atherosclerotic plaque once it has been formed. The principal value of such drugs is prophylactic.

For this reason it might be well for those interested in public health and preventive medicine to consider seriously the problem of early hypertension, particularly in young and middle-aged adults.

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